Demystifying Palmar Midcarpal Instability

Shan Shan Jing, MBBS, BSc (Hons), FRCS, Dip Hand[1] Gemma Smith, FRCS[1]
Subodh Deshmukh, MBBS, MS, MCh, FRCS[1]

[1] Royal Orthopaedic Hospital, The Woodlands, Bristol Road South, Birmingham, United Kingdom

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Address for correspondence Shan Shan Jing, MBBS, BSc(Hons), FRCS, Dip Hand, Department of Hand Surgery, Queen Elizabeth Hospital, Mindelsohn Way, Birmingham B15 2TH, United Kingdom (e-mail: shanshanjing@gmail.com).

Abstract

Palmar midcarpal instability is an uncommon condition diagnosed clinically with a painful pathognomonic clunk on terminal ulnar deviation of the wrist. Various causes have been described, but congenital laxity of the carpal ligaments is thought to be a key contributor. Treatment commences with conservative measures. This includes proprioceptive training based on more recent concepts on the sensorimotor function of the wrist. When these measures plateau, surgery is considered. The lack of high-level evidence and consensus on its cause continue to hamper our understanding and knowledge of this condition. The purpose of this review is to examine the current evidence to conceptualize this mysterious, yet infrequent phenomenon, and to provide an algorithm on its management.

Keywords
- ► midcarpal
- ► instability
- ► chronic
- ► hypermobility
- ► arthrodesis
- ► proprioception

Midcarpal instability (MCI) first described by Mouchet and Belot is an infrequent and poorly understood group of carpal instability disorders.[1] The common feature is a physiological uncoupling of the midcarpal joint in the absence of proximal carpal row dissociation that results in asynchronous motions between the two carpal rows. Lichtman and Wroten grouped MCI into extrinsic and intrinsic instabilities.[2] The intrinsic category is subdivided into palmar, dorsal, and combination types according to the direction of instability. Of these, palmar MCI (PMCI) is the commonest. Extrinsic types are caused by structural abnormalities outside the carpus, most commonly from a distal radius malunion, resulting in adaptive changes within the carpus. Diagnosis is mostly clinical. A high index of suspicion with the support of videofluroscopy and arthroscopic assessments are required when it is absent.[3] Management is aimed at stabilizing the midcarpal joint. Some cases can be self-limiting with conservative treatments. However, refractory cases or static instabilities may require surgery.

Nearly a century on, many questions remain from its etiology to treatment. This stems from a shortage of high-level evidence. To add to the challenge, there is currently no consensus on the definitions and descriptions of MCI and its subtypes. Past clinical studies have assigned confusing terminologies and often contained mixed pathologies in their reporting.[4,5] The focus of this review is to better understand PMCI, by examining the latest evidence on its pathology, biomechanics, and management.

Methods

We searched several databases in December 2019 including PubMed, Cochrane, Elsevier Journals, and SAGE Journals for studies published in English, using Medical Subject Headings (MeSH) terminology, and synonyms for the key terms: "midcarpal instability," "carpal instability," and "wrist hypermobility." Citations from the reference lists of relevant publications and reference textbooks were also reviewed. The list of articles was compared with review articles to ensure completeness. Manuscripts pertaining to PMCI were recorded but other forms of midcarpal or carpal instabilities were excluded. Of a total 1,058 items identified, 66 were selected. Five were book chapters and the rest were original articles. Of these, 17 (25.7%) were review papers.

Results

Wrist Biomechanics

Wrist biomechanics remains a subject of debate. The columnar, link and slider-crank concepts are all proposed theories of how the wrist functions.[2,6]

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Lichtman et al described the ring theory, which is key to understanding PMCI.[7] In a normal wrist, the proximal and distal carpal rows are linked by the scaphotrapeziotrapezoid (STT) and triquetrohamate (TH) joints and move reciprocally. In radial deviation, the proximal row flexes starting at the STT joint and the proximal pole of capitate and hamate tilt palmarly. In ulnar deviation, the proximal row extends starting at the TH joint, while the proximal capitate and hamate tilt dorsally. This mass effect arises from a combination of joint geometry, bony restraints, and dynamic forces acting on the wrist joints during movement. A break or stretch in the "ring" leads to a predictable dissociative or nondissociative deformity, described largely by Dobyns and Linscheid between the 1970s and 1980s.[6] PMCI is considered a type of carpal instability nondissociative (CIND), caused by the loss of longitudinal ligament constraints that lead to hypermobility of proximal row relative to the distal row and radius.[2,4] PMCI, however, also shares features with volar intercalated segmental instability (VISI).

Pathophysiology

In PMCI, the smooth transition of the carpus during radioulnar deviation is lost. Most authors agree that multiple ligaments must fail for this to occur. As a result, the proximal row stays locked in a flexed position until extreme ulnar deviation, when under significant forces focused on the TH joint, the entire proximal row suddenly snaps back and reduces into an extended position taking the distal row with it. This classically is associated with a painful clunk, known as the catchup clunk.[8]

The radioulnar movement of the wrist (especially ulnar deviation) primarily occur at the midcarpal joint with a lesser contribution from the radiocarpal joint.[9] This may explain why PMCI is particularly apparent during this movement. Interestingly, clunking does not occur during the dart-thrower's motion (dorsoradial wrist extension to palmar–ulnar flexion), which is thought to occur primarily at the midcarpal joint.[10] However, Kane et al had shown that the radiocarpal and midcarpal joints can contribute to this vital movement independently by simulating partial wrist fusions.[11] Further kinetic study is required to elucidate the extrinsic or intrinsic factors at play in PMCI. Different wrist kinetics during radioulnar deviation with morphological variants of lunate may also contribute to PMCI in ulnar minus patients.[2,12,13]

Several studies have demonstrated that volar triquetrohamatecapitate (ulnar arm of the arcuate ligament) and dorsal radiocarpal (DRC; also known as the radiolunotriquetral) ligaments (►**Fig. 1**) are involved in PMCI, resulting in dorsal midcarpal subluxation and the catchup clunk.[7,14,15] Others have also implicated damage to additional ligaments on the radial side of midcarpal joint in some subtypes of PMCI to occur.[4,16–18] These include the STT and scaphocapitate ligaments, which separates the proximal and distal row further. This allows the scaphoid to flex, hinging on the intact radioscaphocapitate ligament, taking the proximal row with it. Indeed, Caputo et al classified MCI differently, into four types depending whether the pathology was on the ulnar (types 1 and 2) or radial side (types 3 and 4) of the wrist.[19] Type 3 involves STT ligament laxity. Type 4, the most severe form, is associated with scapholunate ligament disruption. This may explain why some ulnar sided soft-tissue procedures for PMCI have mixed results.[16,19]

Cause

Common cited causes are trauma and congenital laxity of the implicated ligaments. The latter typically presents during adolescence or periods of hormonal changes. These patients are often symptomatic unilaterally. Hence, some authors consider PMCI as an inherent problem, which only become symptomatic after an injury or loss of proprioception.[3,4,20] Up to 70% of Ehlers–Danlos syndrome patients, mostly with the hypermobility type, reportedly have painful midcarpal laxity.[21] This generally tightens up with time. Therefore, in these patients the instability can be self-limiting.[5,21]

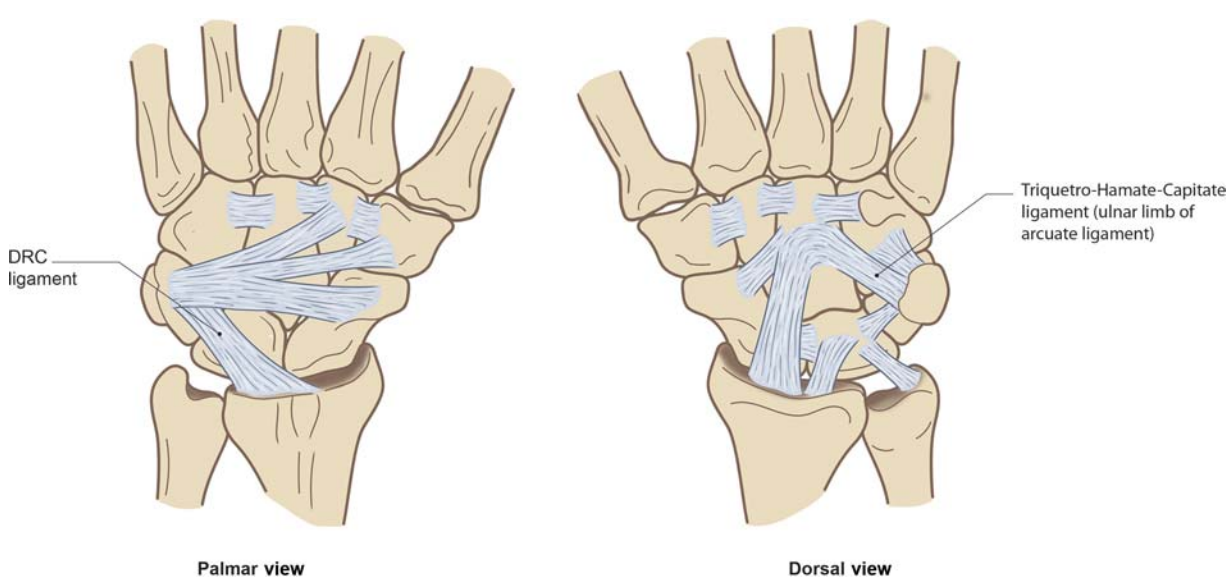

Fig. 1 Ligaments involved in the development of palmar mid carpal instability. DRC, dorsal radiocarpal.

Presentation

The prevalence of PMCI remains unknown, but it is likely to be underestimated because few patients are symptomatic (Chou et al). Typically, the patient is female between 20 and 30 years of age. Only 14 skeletally immature patients with MCI in general have been reported in the literature, mostly during adolescence.[22] Presentation varies from vague ulnar-sided wrist pain that subside with rest to debilitating painful subluxation during activities requiring radioulnar deviation.[3,4,23] It is important to differentiate PMCI from other sources of ulnar-sided wrist pain. Frequently, the onset is insidious. Precipitating trauma if recalled ranges from trivial injuries, to falls or road-traffic accidents that apply a rotational axial force onto an outstretched hand as described by von Schroeder.[23,24] A history of repetitive loading of the wrist may be present.

Physical Examination

Examination findings depend on the severity of PMCI. At rest, there may be volar translocation of the wrist, particularly of the ulnar column and an apparent prominent ulnar head.[23] Localized synovitis can disguise this. An increased pisostyloid distance is indicative of wrist laxity and volar sagging.[3] Pain can be aggravated by palpation of the TH joint, with forearm supination and resisted pronation. It may be alleviated partially by supporting the volar ulnar side of the wrist.[3,25] Patients may spontaneously reproduce a clunk on ulnar deviation, which is often visible and audible to the examiner. This can correct the volar sag, which reappears when the wrist moves back into neutral, with or without another clunk.[23] Wrist flexion and extension are usually normal. In radial sided PMCI, pain occurs over the scaphoid. The Kirk Watson shift test may be positive if the scapholunate ligament is also ruptured.[19]

The midcarpal shift test described by Lichtman et al and Feinstein et al are validated provocation test, performed to diagnose, or exclude PMCI when the patient cannot voluntarily produce the clunk.[8,26] The examiner positions patient's hand in neutral, forearm pronated, then applies a palmar and proximally directed axial force on the distal capitate, while moving the hand from radial to ulnar deviation. If this recreates the catchup clunk and patient's symptoms, the test is positive. Further confirmation comes from eliminating the clunk by pressing the pisiform dorsally on repeating the test.[5,23]

Beighton's hypermobility scores can be recorded to assess generalized ligamental laxity, which may be evident in the nonaffected side.[3,24] Functional evaluations including the disabilities of the arm, shoulder, and the hand (DASH) and patient-rated wrist evaluation (PRWE) can monitor outcomes.[24,27]

Grading

Feinstein et al graded the severity of midcarpal joint laxity from I to V in healthy volunteers and in PMCI (►**Table 1**).[26] The higher the grade, the further into ulnar deviation the clunk occurred. Hargreaves introduced a system incorporating predynamic, dynamic, and static components of PMCI, and proposed management for each (►**Table 2**).[4] PMCI is predynamic if it is asymptomatic, dynamic if patient is symptomatic, and static if a fixed VISI deformity is present.[4] Mason and Hargreaves also used the ability to pour a kettle to infer a subjective measure of instability, and categorized patients according to the frequency of PMCI symptoms while pouring.[28]

Table 1 Lichtman's grading for midcarpal laxity

Grade	Palmar midcarpal translation	Clunk
I	None	None
II	Minimal	Minimal
III	Moderate	Moderate
IV	Maximal	Significant
V	Self-induced	Self-induced

Table 2 Hargreave's grading system for PMCI VISI: volar intercalated segmental instability assessed on lateral radiograph at rest

Grade	Description
0 Presymptomatic	No symptoms but can perform catchup clunk
1 Dynamic	Symptoms of giving way No voluntary clunk or sag Positive midcarpal shift test
2 Voluntary dynamic	Symptomatic giving way with voluntary subluxation
3 Static reducible	VISI deformity reducible on manipulation
4 Static irreducible	Fixed VISI deformity Not easily reducible/locked

Abbreviations: PMCI, palmar midcarpal instability; VISI, volar intercalated segmental instability.

Investigations

Clinical diagnosis remains the gold standard. Standard investigations are frequently normal.[20] Static PMCI may show a VISI deformity (►**Fig. 2**) or widened scapholunate junction on standard radiographical views.[23,24,29] Various angles such as the capitolunate, radiolunate (RL), and lunotriquetral (LT) angles can be measured to define the degree of instability.[30,31] There is no evidence that degenerative changes within the carpus occur secondary to untreated PMCI.[4]

Newer imaging techniques, including three-dimensional computed tomography or 3-tesla (T) magnetic resonance imaging (MRI), are becoming popular noninvasive methods to visualize the carpal morphology and kinematics.[9,32] Videofluoroscopy in different planes enables dynamic assessment of PMCI. It may illustrate the pathognomonic catchup clunk and volar carpal sag. Wrist arthroscopy can rule out pathologies of the intrinsic ligaments and allows for simultaneous thermal capsulorrhaphy if appropriate.[20,28] However, caution should be exercised over positive findings which may not be the source of PMCI.[20]

Fig. 2 Plain radiographs illustrating a volar intercalated segmental instability type deformity.

Management

Majority of PMCIs respond to conservative treatments. Surgery is reserved for refractory cases with the aim to stabilize the midcarpal joint by means of a soft-tissue procedure or limited carpal arthrodesis. Numerous techniques have been described but lack long-term data. Arthrodesis is generally more reliable and offers a solution when soft-tissue stabilization fails.[2,4,33]

Nonsurgical Management

Conservative management begins with understanding the severity and location of PMCI instability. It is most effective for predynamic and dynamic types (Harwood and Turner). Patient education, activity modification, adaptation of workplace ergonomics, and use of simple analgesia are integral part of the treatment to minimize pain or clunking in all cases.[2,34–36]

Basic therapy involves the use of dynamic splints to maintain motion and encourage compliance.[37] The most well-known is the three points of pressure forearm device by Duncan.[38] It applies a dorsal pressure on the pisiform and volar pressure on the ulnar head, to correct the volar sag, and prevent the catchup clunk.[2,34] The suggested duration of splinting is variable. O'Brien supplemented splinting with strengthening exercises, ultrasound and fluidotherapy for 8 weeks.[39] Wong et al advised 3 months or more before weaning off.[37] Six out of 10 and 4 of 7 patients treated by Lichtman et al and Wright et al, respectively, achieved satisfactory outcomes with splints alone, but the regimen was not reported.[7,13] Ho et al successfully treated nine of 16 patients with splintage and proprioceptive training.[3] Three years later, five had no pain, four had reduced pain, and all resumed normal duties. Von Schroeder treated 27 wrists with immobilization for up to 8 weeks, majority of which were static PMCI.[24] In 22 wrists, there was an improvement in pain, but all later relapsed. It maybe that the instability had progressed beyond the point of conservative measures. Ono et al experienced a similar short-term benefit from immobilization with recurrence of pain.[40]

Recent evidence has emerged on the use of proprioceptive and neuromuscular training for PMCI following the success of treating large joint instabilities.[34,40,41] The same is hoped for the wrist, but it is structurally unique as the proximal row lacks tendinous insertion.[34] It is based on the theory that a loss of mechanoreceptors within damaged ligaments leads to deactivation of secondary muscular support, which then manifest as carpal instability.[42] The aim of these treatments is to activate extrinsic stabilizers to substitute for the lax ligaments. Proprioceptive training involves joint position and motion sensing exercises and mirror therapy. Neuromuscular rehabilitation targets flexor and extensor carpi ulnaris and hypothenar muscles cocontractions to drive the pisiform dorsally, and to pronate and extend the proximal carpal row reducing the palmar sag.[16,34,42–44]

Soft-Tissue Stabilization

A variety of soft-tissue procedures have been described based on pathomechanical assumptions. They include open or arthroscopic tightening of the capsule and ligament reconstruction.[4,5] The results are mixed but compared with carpal arthrodesis they may potentially preserve greater motion if successful.[27]

Lichtman et al performed nine soft-tissue procedures in 14 patients: palmar capsulodesis (closing the of the space of Poirer), dorsal capsulodesis (tightens the DRC ligament), advancement of the ulnar arm of arcuate ligament, TH ligament reconstruction with a split ECU tendon, and a combined procedure involving arcuate ligament advancement and DRC capsulodesis.[8] Only three were successful. Comparable to limited carpal fusions in their series, fusions fared better in grip strength and return to function. The relatively poor outcome seen in isolated capsulodesis or ligament advancements may stem from the fact that often multiple ligaments are implicated to produce PMCI.

In milder cases, Ming et al continues to perform dorsal capsulodesis with "promising" results. It reportedly stabilizes the proximal row and eliminates the clunk.[2,23,45] Von Schroeder modified this technique on 27 wrists by narrowing the gap between the dorsal intercarpal and DRC ligaments along the fifth extensor compartment. This stabilized the midcarpal joint and proximal row.[24] After a mean follow-up of 35 months, patients had improved pain, grip strength (range: 19–32 kg) and PRWE (range: 74–37). Two patients had recurrent PMCI and three had chronic pain unrelated to PMCI. The wrist flexion reduced, but other functions were preserved. The best results were observed in females under 25 years with hypermobility, while men over 25 years with a history of moderate–to-severe trauma performed the worst.

Several authors attempted to strengthen the TH junction, considered the "weak link." Early efforts using a dorsal tendon sling between the triquetrum and hamate failed.[4,46,47] Ritt and de Groot performed extensor carpi radialis brevis (ECRB) transposition fixed to the hamate in 13 dynamic cases with early active mobilization.[48] They achieved 11 good outcomes and two failures in a follow-up up to 6 years. Chaudhry et al reconstructed the dorsal TH ligament in seven wrists using a palmaris longus graft anchored in a zig-zag fashion between the triquetrum and hamate.[27] There was an overall improvement in grip strength and DASH score (49–28) with a mean follow-up of 28 months. Patients lost some ulnar deviation and flexion but retained other wrist functions. There were three failures from recurrent instability and pain. Garcia-Elias described the use of ECRB to reconstruct triquetral-hamate-capitate and DRC ligaments but the outcome is unknown.[16]

Arthroscopic thermal capsular shrinkage has enjoyed some success in milder cases of PMCI. Mason and Hargreaves published encouraging results in 15 wrists.[28] All had an improvement in their instability and DASH score with a small reduction in wrist motion. After 10 years, the same cohort showed no significant deterioration.[4] Ho et al had less favorable results.[3] They treated five of seven patients targeting all but the DRC ligament using this technique to minimize complications. Two had recurrent instability corrected by DRC ligament reconstruction using a strip of flexor carpi ulnaris tendon along with three additional patients. The tendon strip was redirected from palmar to dorsal through the triquetrum, then fixed to Lister's tubercle. This imparted an extended and pronated torque on the proximal carpal row reducing the VISI deformity. At the final follow-up averaging 86 months, all five patients had improved grip strength (67–82% of the contralateral side) and in the wrist performance score and resumed their original work. Three had complete pain relief. One had mild pain and another had moderate, fluctuating pain.

Zijlker et al recently adopted a more aggressive approach on 24 wrists using a strip of flexor carpi radialis tendon to stabilize the STT joint and imbricate the DRC and DIC ligaments.[49] Authors did not clarify the type of MCI in this cohort. After a mean follow-up of 18 months, 18 patients were satisfied and 14 of 15 returned to work. There were seven revisions including wrist denervation and RL arthrodesis.

Krijgh et al treated five Ehlers–Danlos syndrome patients with MCI with a catchup clunk by looping a strip of ECRB tendon around the lunocapitate joint.[50] The authors felt that capsular procedures would loosen in these patients, and carpal arthrodesis would strain the capsule of adjacent joints. Three were successful in reducing pain and instability which led to less reliance on orthotics on short-term follow-up. One recurrence with postoperative VISI was revised by restoring the lunocapitate alignment and capsulodesis.

Few pediatric cases have been reported. Chou et al treated a posttraumatic PMCI in a 6-year-old with palmar and dorsal capsulodesis imbricating the key ligaments and Kirschner's wiring across the midcarpal joint.[22] After 8 years, the patient had intermittent mild aches only and maintained a reasonable range of movement. The combined soft-tissue procedure avoids the morbidly and long-term sequelae of an arthrodesis in pediatric patients. Wright et al used soft-tissue reconstruction in seven patients under 17 years with a mixed type of MCI, and the result was fair.[13] Mason and Hargreaves treated one patient under 21 years, who had a subjective improvement in PMCI and DASH score at 45 months.[28] Comparatively, Rao and Culver treated two children by limited fusion. Both had nonunion.[25]

Bone Procedures

Isolated TH fusion has had limited success. Lichtman et al performed TH arthrodesis on three patients.[8] Although fully united, two patients developed crepitus and clicking over dorsoradial part of wrist on long-term follow-up. The authors hypothesized that fusion on the ulnar side of the midcarpal joint may lead to a compensatory increase in radial sided mobility. Four-corner fusion is now their preferred treatment. Rao and Culver performed 11 TH fusion with an average follow-up of 26 months.[25] While fusion eliminated clunking, only seven were pain free. One had symptomatic nonunion. The range of motion averaged 55 to 69% compared with the contralateral side and grip strength averaged 64%. Two patients had excellent, four good, three fair, and two poor results based on their postoperative pain, grip, motion, and appearance on radiographs.

Halikis et al performed RL arthrodesis on five dynamic PMCI.[51] It stabilized the proximal row and preserved midcarpal mobility including the dart-throwing motion. One patient developed a nonunion, while another had chronic ulnar-sided wrist pain. Shiga et al replicated PMCI on nine cadavers and simulated TH and RL arthrodesis.[18] The authors found that although both fusions had eliminated the catchup clunk, TH fusion altered the proximal row's motion (increased scaphoid flexion and reduced extension during radioulnar deviation), while RL fusion reduced it. This supports the hypothesis by Lichtman et al that TH fusion can lead to a compensatory hypermobility on the radial midcarpal joint.[8,18]

Four-corner fusion appears to have the best result.[45] In the series by Goldfarb et al, among eight patients with an average follow-up of 34 months, the majority were satisfied with surgery. Six had no or mild pain.[52] One with chronic pain had total wrist fusion. Although the authors noted a significantly reduction in the flexion–extension arc, from 135 to

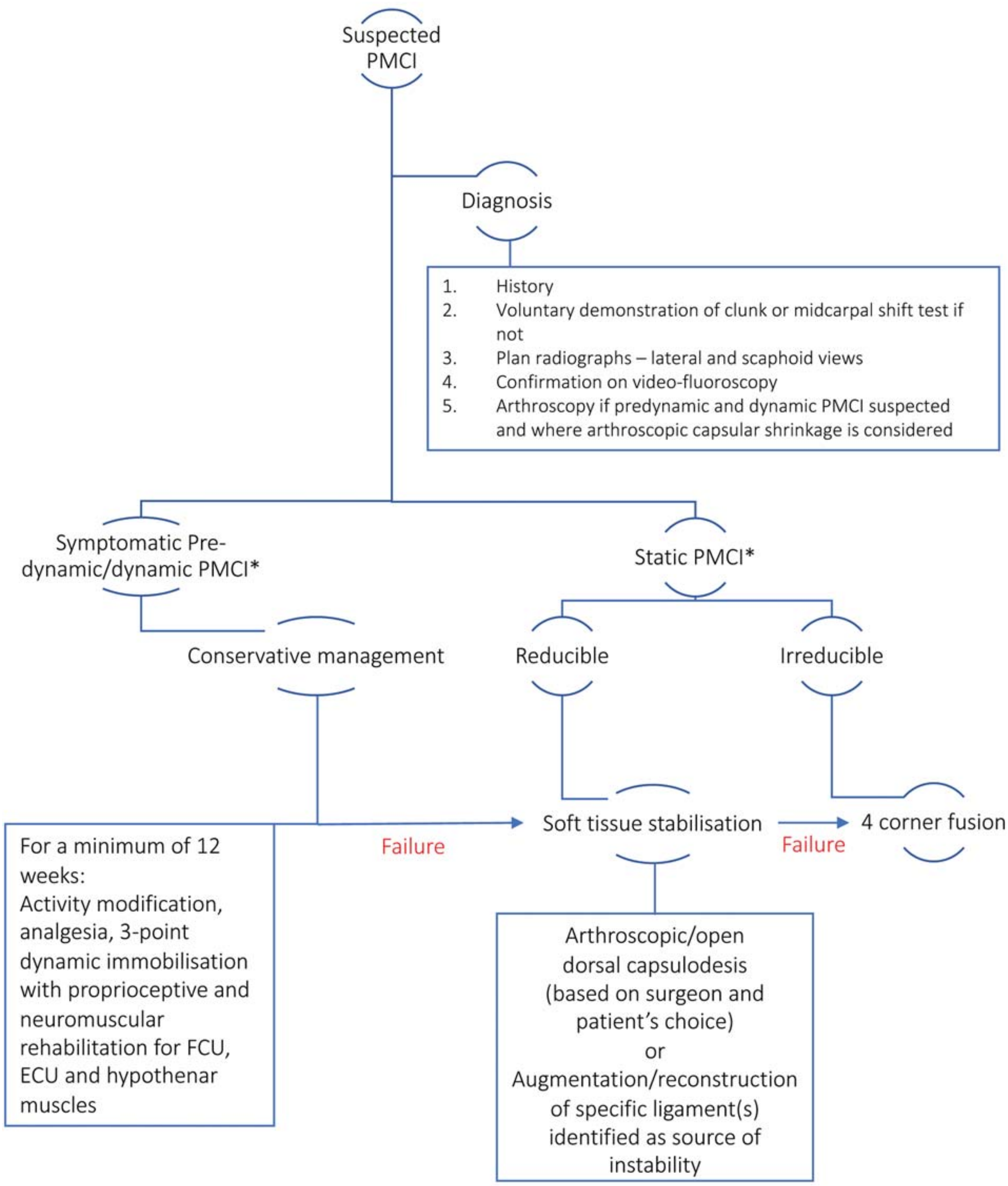

Fig. 3 An algorithm of managing palmar mid carpal instability. ECU, extensor carpi ulnaris; FCU, flexor carpi ulnaris; PMCI, palmar midcarpal instability. * Please refer ►**Table 2**, Hargreaves' grading system for PMCI.

75 degrees, the average grip strength increased from 20 to 32 kg. Proximal row carpectomy has also been described in a four-case series.[4] This resolved the instability, but patients lost 50% of their movement. The risk of long-term degenerative arthritis following four-corner fusions and proximal row carpectomy for PMCI remains unknown.[4] Caputo et al has recommended STT fusion for radial-sided PMCI. Although conceptually sound, clinical data are lacking.[19]

Discussion

PMCI remains poorly understood. This is complicated by the use of confusing nomenclatures and a sparsity of high-level evidence.[18] Most reports are based on small retrospective case series with a short follow-up and a lack of diagnosis and severity of the type of MCI being treated. Definitions of outcomes also varied. Failure may include persistent pain, recurrence of deformity, inability to return to activities, or complications related to management. Collectively, they cloud the assessment and interpretation of results.

Typically, patients are young women with minor trauma and chronic ulnar-sided wrist pain. Clinical diagnosis remains the gold-standard illustrating a painful catchup clunk near the end of ulnar deviation. It can be confirmed on videofluoroscopy. An MRI may rule out other wrist abnormalities. Management depends on the correct classification and grading of PMCI. First-line treatment is conservative, including splintage and proprioceptive and neuromuscular rehabilitation. The focus is to achieve a pain-free stable joint rather than to improve the range of movements.[34] Some cases are self-limiting. Surgery is considered when conservative measures fail. There is no consensus on the ideal intervention. Numerous soft-tissue stabilizations have been described based on suspected pathologies with mixed results. They involve either (1) dorsal soft-tissue stabilization to augment or reconstruct the DRC or TH ligament or (2) volar soft-tissue stabilization by palmar capsulodesis or advancing the ulnar arm of arcuate ligament. Arthroscopic thermal capsular shrinkage can be attempted first, especially in mild cases. However, the long-term benefit of these procedures remains unknown. Several fusion options have been described. However, four-corner fusion appears to be more reliable but is not without its complications. Arthrodesis in younger patients require careful counselling, as it can significantly reduce movements and alter wrist biomechanics with downstream effects.

Limitations

A limitation of this review is that only articles published in English are analyzed. There are many questions that remain unanswered, including the relative importance of volar and dorsal ligaments in the causation of PMCI and natural history of the disease progression, particularly in normal wrist variants and in hypermobile patients. The point at which global ligament laxity becomes pathological is also undetermined. Quality prospective studies with large sample sizes and objective diagnostic criteria (ulnar or radial instability or mixed type) are required. Without a better understanding of its pathomechanics and a systematic method of evaluation, it is difficult to offer the appropriate management or predict outcomes.

Conclusion

This review summarizes our current understanding of PMCI based on the most recent evidence. From this and our experience, we suggest that diagnosis of PMCI is largely clinical through a thorough history and detailed examination followed by appropriate investigations. Treatment options are advised once the grade and type of PMCI are established. Proprioceptive and neuromuscular training has advanced the treatment of predynamic and dynamic cases. Surgery is considered if therapy fails after a minimum period of 3 months. We advise that dorsal capsulodesis should be attempted first, given its superior results. Bony procedures are reserved for recalcitrant static cases after careful patient counselling. Of these, four corner fusion without scaphoidectomy is preferred. We have distilled this into a management algorithm (►**Fig. 3**). Overall, the prognosis of all these procedures remains guarded as the long-term results are largely unknown. Such is the challenge of this rare condition. Although better recognized, further research is required to define the pathogenesis of PMCI. Sufficiently powered trials are required to provide meaningful results and validation of various treatments.

Authors' Contributions

S.S.J. acquired, analyzed, and interpreted majority of the data and primary contributor to the manuscript including its concept and design. G.S. reviewed 10 articles and revised the manuscript critically. S.D. is the senior author who provided intellectual support and revised the manuscript critically.

Ethical Approval

The authors declare that no ethical approval was required for this article. Also, no informed consent was required for this article.

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Conflict of Interest

None declared.

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